Correspondence

Seborrheic dermatitis in COVID-19: a case report

Dear Editor,

This report analyzes the case of a 63-year-old Caucasian male, Fitzpatrick III phototype, who was admitted to the Intensive Care Unit (ICU) due to a severe coronavirus disease 2019 (COVID-19) pneumonia. The patient's medical history was remarkable for valvular heart disease, the presence of a biological aortic valve, and primary arterial hypertension, but he had no prior history of any dermatological complaints. The patient was chronically medicated with furosemide 40mg bid, apixaban 5mg bid, and amlodipine 5mg od. At admission, laboratory studies revealed mild lymphopenia (900/μL). Lopinavir/ritonavir and hydroxychloroquine were administered for 14 days. He was ventilated at admission for 21 days until his respiratory function improved.

At day 33, the patient developed a nonpruriginous erythematosquamous dermatosis localized in the face, affecting the forehead, supraciliary regions, and nasolabial folds, with outward extension to both cheeks (Fig. 1). The scale was coarse and adherent. The patient denied any previous episodes, and no other changes were detected on dermatological examination. Seborrheic dermatitis (SD) was diagnosed, and the patient was prescribed hydrocortisone cream bid, with complete resolution of the dermatosis after 7 days.

Several dermatological manifestations of COVID-19 have been described,[1] but no reports of SD in COVID-19 have been published so far. At our center, we have seen several cases of SD in patients admitted to the ICU due to COVID-19; the association between the two diseases may not be incidental. While the pathophysiology of SD is not consensual, cellular immunity, inflammatory cytokines, and neurogenic factors have been implicated in its pathogenesis.

Severe COVID-19 may be seen with lymphopenia which in turn may be a contributing factor to SD onset, similar to individuals with the human immunodeficiency virus (HIV) infection, and particularly those with CD4 + counts in the 200-500/μL range, show increased incidence and severity of SD.[2] The onset of a severe form of SD is often the sign which leads to the diagnosis of HIV.

Increased production of inflammatory cytokines may also link COVID-19 to SD. It is now accepted that the most severe forms of COVID-19 are caused by a "cytokine storm" and not by a direct cytopathic effect of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). This proinflammatory status features high levels of circulating cytokines such as Interleukin-1, Interleukin-6, and tumor necrosis factor alpha (TNF-α). These cytokines have been shown to be present at higher concentration in SD-affected skin than in healthy skin.[3] An immune reconstitution syndrome in HIV infection under antiretroviral treatment is also positively associated with SD; this supports the role of an inflammatory response to the pathogenesis of this dermatosis.

Figure 1 Erythematosquamous dermatosis with thick adherent scale, affecting the forehead, supraciliary regions, and nasolabial folds, with outward extension to both cheeks. These findings are characteristic of seborrheic dermatitis

The third link between the two conditions is neurogenic mechanisms. SARS-CoV-2 shows neurotropism, and neurological manifestations of COVID-19 have been described.[4] Concomitantly, the role of neurogenic factors in relation to SD has been mentioned since patients with neurodegenerative conditions, such as Alzheimer's disease or Parkinson's disease, have increased incidence of SD. The report of significant improvement of SD after stellate ganglion block, which may relate to a

decrease in Interleukin-6 production, provides further support to this hypothesis.[5]

SD and COVID-19 may share common mechanisms, which could explain cases such as the one in this report. Awareness about this dermatosis is necessary to prevent a misdiagnosis of contact dermatitis to either medical devices or personal protective equipment. When assessing patients with *de novo* onset of severe SD, or significant exacerbation of a previously mild SD, physicians should consider the possibility of underlying COVID-19 as they do for HIV infection. Such cases require a thorough anamnesis and epidemiological survey, rather than preemptive testing in all cases, as other factors may lead to the onset or aggravation of SD (e.g., prolonged use of PPE).

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The patient in this manuscript has given written informed consent to the publication of his case details.

Miguel Alpalhão[1,2,3]*, MD
Nuno Gaibino[4], MD
Paulo Filipe[1,2,3], MD, PhD

[1]Dermatology and Venereology Department, Hospital de Santa Maria, Centro Hospitalar Universitário Lisboa Norte EPE, Lisbon, Portugal
[2]Dermatology and Venereology University Clinic Faculty of Medicine, University of Lisbon, Lisbon, Portugal
[3]Dermatology Research Unit, iMM João Lobo Antunes, University of Lisbon, Lisbon, Portugal
[4]Intensive Care Department, Hospital de Santa Maria, Centro Hospitalar Universitário Lisboa Norte EPE, Lisbon, Portugal
*E-mail: migueldbalpalhao@campus.ul.pt

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